WE CLAIM:

1. A method for treating a subject having, or susceptible to having, a type I hypersensitivity, asthma or an allergy comprising administering a therapeutically effective amount of at least one PPAR-γ agonist, or derivative thereof, to said subject, wherein said administration of said at least one PPAR-γ agonist, or derivative thereof, is effective to treat said type I hypersensitivity, asthma or allergy in said subject.

2. The method of claim 1 wherein said PPAR-γ agonist is selected from the group consisting of a thiazolidinedione and a non- thiazolidinedione PPAR-γ agonist.

3. The method of claim 1 wherein said at least one PPARγ agonist is selected from the group consisting of Ciglitazone, Troglitazone, Rosiglitazone, Pioglitazone, Englitazone, RXR activator LGD1069, and prostaglandin J2.

4. The method of claim 1, wherein said subject has, or is susceptible to having, an asthma.

5. The method of claim 4, wherein said asthma is allergic asthma.

6. The method of claim 1, wherein said therapeutically effective amount of said PPAR-γ agonist is approximately from 2 mg/kg to 10 mg/kg per day.

7. The method of claim 1, wherein said therapeutically effective amount of said PPAR-γ agonist is approximately 2 mg/kg per day.

8. The method of claim 1, wherein said administering is selected from the group consisting of aerosol, parenteral, oral, intravenous, intramuscular, intraperitoneal, transdermal, rectal, buccal and subcutaneous administering.

9. The method of claim 1, wherein said subject is a mammal.

10. The method of claim 9, wherein said mammal is human.

11. The method of claim 2, wherein said PPAR-γ agonist is a thiazolidinedione derivative.

12. The method of claim 11, wherein said thiazolidinedione derivative is administered by a route selected from the group consisting of aerosol, parenteral, oral, intravenous, intramuscular, intraperitoneal, transdermal, rectal, buccal and subcutaneous administration.

13. The method of claim 11, wherein said thiazolidinedione derivative comprises a thiazolidinedone-2 derivative or a 4-diketone substituted derivative.

14. The method of claim 11, wherein said therapeutically effective amount of said thiazolidinedione derivative is approximately 2 mg/kg to 10 mg/kg per day.

15. The method of claim 11, wherein said therapeutically effective amount of said thiazolidinedione derivative is approximately 2 mg/kg per day.

16. The method of claim 11, wherein said subject is a mammal.

17. The method of claim 16, wherein said mammal is a human.

18. The method of claim 2, wherein said PPAR-γ agonist is a non-thiazolidinedione PPAR-γ agonist.

19. The method of claim 18, wherein said non-thiazolidinedione PPAR-γ agonist. is administered by a route selected from the group consisting of aerosol, parenteral, oral, intravenous, intramuscular, intraperitoneal, transdermal, rectal, buccal and subcutaneous administration.

20. The method of claim 18, wherein said non-thiazolidinedione PPAR-γ agonist comprises a piperazine or heterocycle derivative.

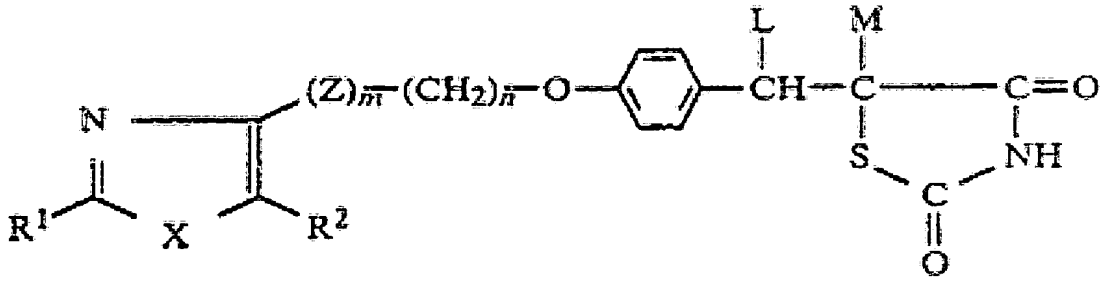
21. The method of claim 18, wherein said therapeutically effective amount of said non-thiazolidinedione PPAR-γ agonist is approximately 2 mg/kg to 10 mg/kg per day.

22. The method of claim 18, wherein said therapeutically effective amount of said non-thiazolidinedione PPAR-γ agonist is approximately 2 mg/kg per day.

23. The method of claim 18, wherein said subject is a mammal.

24. The method of claim 23, wherein said mammal is a human.

25. A method for treating a subject having, or susceptible to having, a type I hypersensitivity, asthma or allergy, comprising administering to said subject a therapeutically effective amount of a compound comprising Formula I:



wherein R^1 is hydrogen, hydrocarbon residue, or heterocyclic residue which may each be substituted;
R^2 is hydrogen or lower alkyl which may be substituted by a hydroxyl group;
X is an oxygen or sulfur atom;
Z is a hydroxylated methylene or carbonyl;
m is a value of 0 or 1;
n is an integer having a value of from 1 to 3; and
L and M combine with each other and cooperate jointly to form a linkage and a plurality of salts.

26. The method of claim 25, wherein said subject has, or is susceptible to having, an asthma.

27. The method of claim 26, wherein said asthma is allergic asthma.

28. The method of claim 25, wherein said therapeutically effective amount of said compound is approximately from 2 mg/kg to 10 mg/kg per day.

29. The method of claim 25, wherein said therapeutically effective amount of said PPAR-γ agonist is approximately 2 mg/kg per day.

30. The method of claim 25, wherein said administering is selected from the group consisting of aerosol, parenteral, oral, intravenous, intramuscular, intraperitoneal, transdermal, rectal, buccal, or subcutaneous administration.

31. The method of claim 25, wherein said subject is a mammal.

32. The method of claim 31, wherein said mammal is a human.

33. An *in vivo* method of identifying a compound effective to treat type I hypersensitivity, asthma or allergy in a subject comprising:

a) contacting a group of one or more subjects with a test compound to form a first population;

b) contacting a different group of one or more subjects with a PPAR-γ agonist to form a second population;

c) inducing type I hypersensitivity, asthma or said allergy in said first and second populations; and,

d) comparing one or more symptoms of said type I hypersensitivity, asthma or allergy in said first and second populations;

wherein when said one or more symptoms of said type I hypersensitivity, asthma or allergy in said first population is less than or the same as said one or more symptoms

of said type I hypersensitivity, asthma or allergy in said second population, a compound effective to treat type I hypersensitivity, asthma or allergy in a subject is identified.

34. The method of claim 33, wherein said one or more symptoms is selected from the group consisting of an increase in T_H2 type cytokines, lung airway inflammation, eosinophil infiltration, mucous production in the lung, airway hyperreactivity (AHR) and elevated serum IgE levels.

35. The method of claim 33, wherein said subject is a mammal.

36. The method of claim 35, wherein said mammal is human.

37. The method of claim 33, wherein said asthma is allergic asthma.

38. A compound identified by the method of claim 33.

39. The compound of claim 38 in a pharmaceutically acceptable carrier.

40. The method of claim 33, wherein said agonist is Ciglitazone.

41. A method of regulating T_H2 cell function in the lung airway of a subject in need of said regulating comprising administering to said subject an amount of a PPAR-γ agonist effective to regulate said T_H2 cell function in said lung airway of said subject.

42. The method of claim 41, wherein said T_H2 cell function is selected from the group consisting of T_H2 cell cytokine production, inflammation, eosinophil infiltration, mucous production, airway hyperreactivity and epithelial cell thickening.

43. The method of claim 42, wherein said T_H2 cell cytokine production comprises production of IL-4, IL-5 and IL-13.

44. An in vitro method for identifying a compound effective to treat type I hypersensitivity, asthma or allergy in a subject comprising:

a) culturing a first T cell population under T_H2 priming conditions to obtain a primed first cell population;

b) culturing a second T cell population under T_H2 priming conditions to obtain a primed second cell population;

c) stimulating said first primed cell population with a PPARγ agonist;

d) stimulating said second primed cell population with said test compound; and,

e) comparing the amount of secretion of one or more cytokines from said cell populations in part c) and part d);

wherein when the cytokine secretion from said cell population of part d) is less than or equal to the cytokine secretion from the cell population of part c), a compound effective to treat type I hypersensitivity, asthma or allergy in a subject is identified.

45. The method of claim 44, wherein said PPARγ agonist is Ciglitazone.

46. The method of claim 44, wherein said one or more cytokines is selected from the group consisting of IL-2, IL-5, IL-13 and IFNγ.

47. The method of claim 44, wherein said subject is a mammal.

48. The method of claim 47, wherein said mammal is human.

49. A compound identified by the method of claim 44.